

Since the middle of the eighteenth century, when Von Rosenstein first advocated the dilution of cow's or goat's milk with water or cereal water to make it more digestible for the infant, many other methods of modifying milk have been advocated. Boiling, evaporating, powdering, peptonizing, the addition of alkalis or acids, the removal of whey, the removal or addition of cream, the addition of various cereals and various sugars are the most common modifications. Assuming that we understand how and when to employ these various modifications and using all our present knowledge in prescribing feedings, still we frequently see babies with colic. Ruling out surgical conditions, focal or systemic infection and improper nursing care, I believe these cases can nearly always be shown to be allergic and when handled as such the results are often most gratifying.

It is known that boiled or evaporated milk forms a smaller and softer curd than raw milk, and this has been the explanation for the fact that babies have less feeding disturbances when cooked milk is given. Perhaps there is also another explanation. According to Freudenberg, the lactalbumin passes through the intestinal wall easily and cooking diminishes this absorption and the possibility of sensitization. Cutter, in studying the antigenic properties of evaporated milk, reports a diminution in the whey protein and no change in the casein. It seems possible that some of our good results with *eiwissmilch* or protein milk may be thus accounted for, also.

Considerable has been written in the past few years about the rôle of infection in some of these troublesome feeding cases, but practically nothing about allergy, and I believe this paper of Doctor Stafford's brings out a new chapter in infant feeding. Even the latest textbooks on pediatrics and infant nutrition have apparently overlooked this most important subject.

Everyone who is feeding a large number of babies must agree that colic is not uncommon. That frequently no apparent cause can be found. That by removing or altering one or more of the protein elements in the formula the symptoms subside or are relieved. The authors have been very conservative in their conclusions and have not given the impression that all distress in infants is due to abdominal allergy.

THE CLINICAL CLASSIFICATION OF PULMONARY TUBERCULOSIS*

By SIDNEY J. SHIPMAN, M. D.
San Francisco

DISCUSSION by Chesley Bush, M. D., Livermore; F. M. Pottenger, M. D., Monrovia; C. E. Atkinson, M. D., Banning.

THE older accepted classifications of pulmonary tuberculosis, such as the Turban classification and the classification of the American Sanatorium Association, have been based largely upon the extent of lesions. Thus, it is customary to speak of minimal, moderately advanced or far advanced lesions, and, more recently, to add A, B, or C, to denote the severity of the process or symptomatology. Admirable and necessary as is such classification, it is doubtful if it fully covers the ground as far as clinical need is concerned, for the gravity of tuberculosis may be determined no less by the type of pathology than by the extent of actual lesion.

OTHER CLINICAL CLASSIFICATIONS OF PULMONARY TUBERCULOSIS

Fraenkel in 1904 and Albrecht in 1907 attempted to separate pulmonary tuberculosis into two types—the exudative and proliferative. This classification was adopted by Aschoff and his school at Freiburg and has since been accepted by a growing number of clinicians. Siegfried Gräff and Leopold Küpferle¹ in their excellent work, *Die Lungenphthise*, which appeared in 1923, did much to further the popularity of the classification.

Nevertheless this division of pulmonary tuberculosis into two predominant clinical types has encountered some opposition. F. M. Pottenger, in particular, has taken exception to the terms, which in effect separate “into two distinct groups phenomena which are qualitatively the same, differing only quantitatively.”² In the same article Pottenger states:

“The exudative and the proliferative processes, as they are encountered in chronic tuberculosis, are not totally different processes, as would be required in order to make them bases of types into which tuberculosis is to be divided. On the contrary, as we meet them, either in primary tubercle or in the early re-inoculations, whether caused by bacilli escaping from foci in which they have been confined within the body of the host or by bacilli entering from without, or in the course of clinical tuberculosis, they represent different phases of the same tissue reaction to bacillary invasion. The difference in the degree of reaction varies with the degree of specific resistance present and the dosage of bacilli; a few bacilli causing a slight hyperemia with little exudation and the proliferation of fixed cells; while many cause severe inflammatory and exudative phenomena.”

Although denying that exudation and proliferation may be legitimately used as a basis of classification in pulmonary tuberculosis, Pottenger none the less recognizes that they exist, for he discusses them, rightly pointing out that they merely represent different phases of the allergic reaction. Few would quarrel with this statement. Clinical studies carried out on human material, if continued for a sufficient length of time, will show this, as has been demonstrated by Elizabeth Davis and myself.³ In a clinical study, now in its fifth year, of the nurses at the University of California, we have observed the development of clinical pulmonary tuberculosis in previously healthy and roentgenographically negative young women, and we have been struck with the fact that apical proliferative tuberculosis was present concomitantly with moderate skin hypersensitiveness, while exudative lesions have appeared only in the presence of marked cutaneous tuberculin reactions. Skin tests were done both before and after the appearance of manifest tuberculosis, so that we had some idea of the degree of allergy in these young women before they became ill.

What should be emphasized in the exudative-proliferative classification is that it is a classification of clinical expediency only, and that it does not deny the essential unity of tubercle or the part played by allergy in the formation of a lesion. Indeed it is doubtful if any clinician could successfully treat tuberculosis without an under-

* From the department of medicine, University of California Medical School.

* Read before the General Medicine Section of the California Medical Association at the fifty-ninth annual session at Del Monte, April 28 to May 1, 1930.

TABLE 1.—*Clinical Differences in Exudative and Proliferative Lesions*

	Onset	Breakdown	Course	Cavitation	Treatment
Exudative	Usually catarrhal, mild or severe	Recent	Favorable—resolution Unfavorable—rapid extension	Early with thin walls	Rigid
Proliferative	Usually insidious	More remote	Favorable—static lesion Unfavorable—slowly progressive	Late with fibrous walls	A modified life

standing of the part played by allergy; certainly he could not secure the results otherwise obtainable. Without such knowledge the classification itself would be meaningless.

PROLIFERATIVE AND EXUDATIVE GROUPS

Unfortunately not all cases can be classified into either proliferative or exudative groups; in addition, exudative cases become proliferative as time goes on and proliferative cases at times develop fresh exudative lesions. Nevertheless a large number of cases are predominantly proliferative or predominantly exudative and as such may be classified to advantage. Such classification is useful from a clinical standpoint for the following reasons: (1) It is descriptive, and gives some idea of the underlying pathology in the lung; (2) it foretells the possible or probable course of the disease; and (3) it furnishes a valuable guide to treatment. The chief clinical differences in the two types of lesion are noted in the above table.

While the mode of onset in the exudative type is usually catarrhal, the initial symptoms may be so slight as to pass unnoticed, and the event which calls the disease to the patient's attention may be hemoptysis or pleurisy. When detected early there may be nothing except the roentgenographic evidence to indicate the true nature of the disease, for all other symptoms may be lacking. Since the lesion is almost always subapical, physical signs are frequently difficult and sometimes are impossible to detect. The asymptomatic nature of

the disease at this stage is misleading, and unless one recognizes the fact that the lesion is treacherous and is apt to extend readily and to cavitate quickly, a false feeling of security might lead rapidly to disaster.

However, the widespread opinion that the exudative type of pulmonary tuberculosis is inherently an unfavorable type seems to me to be without scientific foundation. Such a supposition ignores the fact that exudation is the result of a high degree of allergy, and we have excellent reasons for believing that allergy and resistance travel hand in hand. Early exudative lesions can heal without clinical trace. Thus, if one ever secures a perfect result in the treatment of pulmonary tuberculosis, it should be secured in these cases. The treatment must be rigid, however. Bed rest accomplishes wonders.

The insidious onset of proliferative lesions usually allows them to become more extensive before detection, although, being usually apical, they can more often be detected by means of physical examination. They can be followed more accurately in the same manner. It is my feeling that these patients can often be successfully treated by a modification of activity if necessary, although in the presence of fever bed rest is, of course, essential.

Surely there is today sufficient clinical advantage in dividing cases of pulmonary tuberculosis into predominantly exudative or predominantly proliferative to make the division useful. For several years the writer has been in the habit of

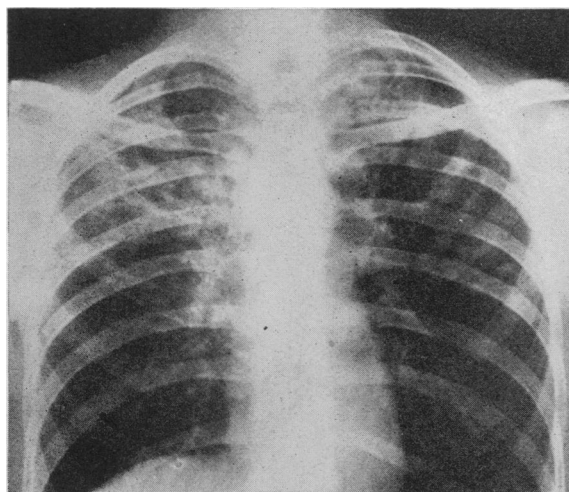


Fig. 1.—Acinous exudative tuberculosis in a young man with laryngeal tuberculosis, who later died with tuberculous meningitis. Note fine distribution of lesion.

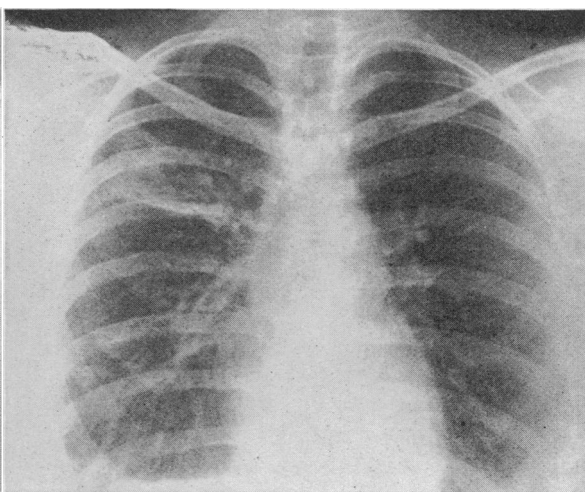


Fig. 2.—Early exudative lesion in the lower part of the right upper, with cavitation behind the second rib. Hemoptoic onset in a girl of nineteen. After initial hemoptysis entirely asymptomatic.

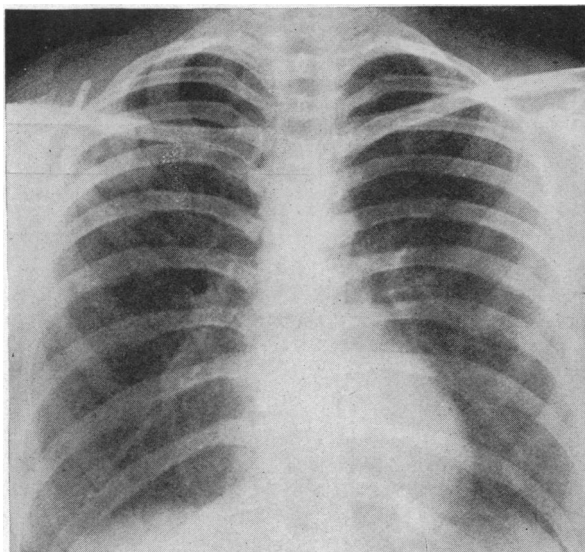


Fig. 3.—Exudative lesion in the right upper with early, fresh cavitation, in a young woman of twenty. Catarrhal onset with symptoms of brief duration. During strict bed rest, cavity enlarged. Clinical cure with compression.

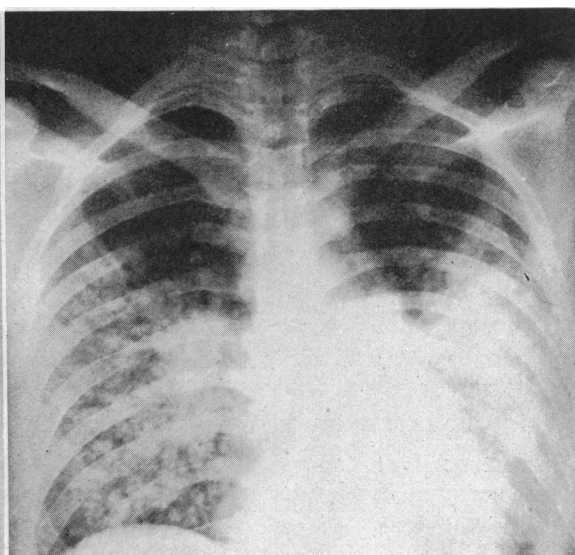


Fig. 4.—T. S. (1-15-27). Widespread lobular exudative extension following intractable hemoptysis. With the onset of hemoptysis the left lung was immediately compressed, and an excellent compression obtained, without, however, any effect upon the bleeding. With acute extension and resultant dyspnea, pneumothorax was abandoned. A small amount of air can be seen in the left pleural cavity. Death occurred two weeks later.

classifying his cases, where possible, into minimal (exudative or proliferative) A, B, and C; moderately advanced (exudative or proliferative), A, B, and C, and so on. While slightly more cumbersome, the classification becomes much more valuable and descriptive. Certainly in this manner it is possible to visualize the character as well as the extent of the lesion.

A number of illustrative plates follow. For a more complete description with excellent illustrations of pathological material, the work of Gräff and Küpferle¹ is recommended.

490 Post Street.

REFERENCES

1. Gräff, Siegfried and Küpferle, Leopold. *Die Lungenphthise. Ergebn. vergleichender Roentgenologisch-Anatomischer Untersuchungen.* 1923.
2. Pottenger, F. M. Exudative and Proliferative Processes not a Basis for Classifying Tuberculosis into Types. *Am. Rev. Tuberc.*, Vol. xviii, No. 5, p. 580.
3. Shipman, Sidney J., and Davis, Elizabeth A. *Tuberculin Sensitiveness and Tuberculous Disease.* *Am. J. Nursing*, p. 769, August 1928.

DISCUSSION

CHESLEY BUSH, M. D. (Arroyo Sanatorium, Livermore).—Doctor Shipman has pointed out the inherent

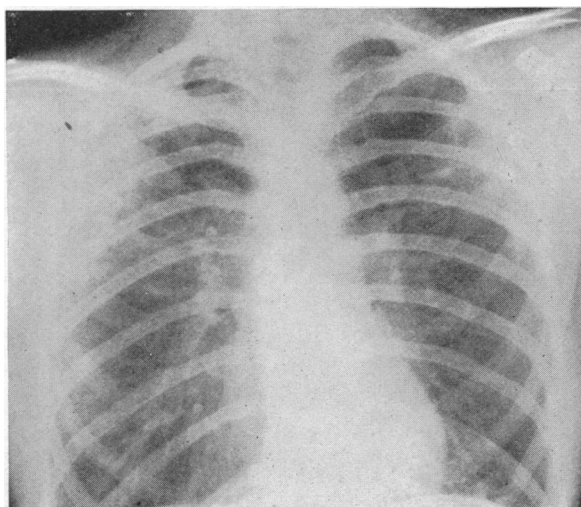


Fig. 5.—A predominantly proliferative lesion, involving both uppers and the left lower, in a woman of twenty-eight. Insidious onset with febrile course for several months. Although the clinical course was satisfactory and the young woman is now pursuing a normal life, the character and extent of the lesion have shown no change in two years.

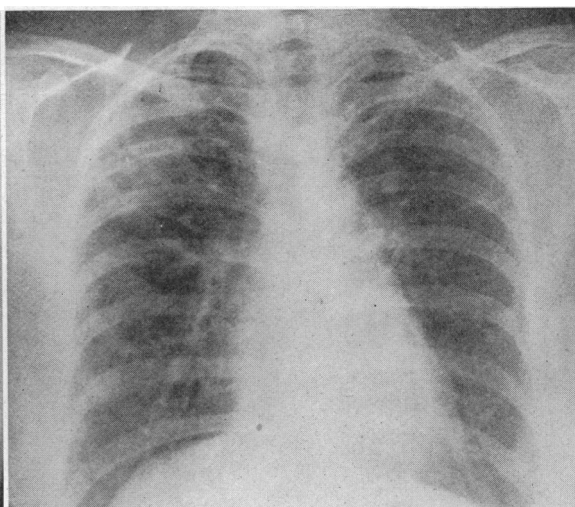


Fig. 6.—A predominantly proliferative lesion, involving chiefly the right upper but distributed throughout both lungs, in a woman of forty-five. Insidious onset with mildly febrile course for several months. For the past five years this woman has been leading a normal life, but the character and extent of the lesion, as far as x-ray evidence is concerned, have not changed since she was first seen, six years ago.

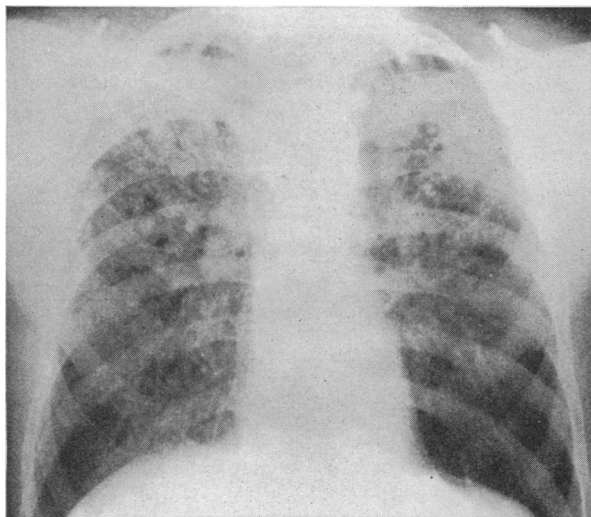


Fig. 7.—Proliferative (fibroid) lesion in a man of forty-six. Insidious onset with mildly febrile course for a few weeks. Marked gain in weight. Clinically well and at work for one year, but for two years the character and extent of the lesion has remained almost unchanged.

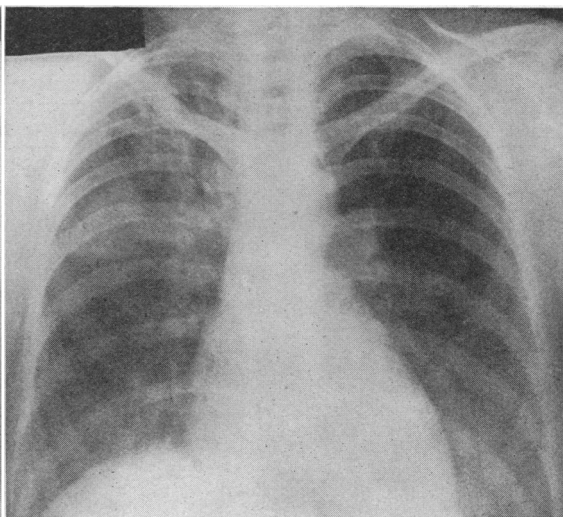


Fig. 8.—Predominantly proliferative and predominantly exudative lesion in a woman of thirty. The proliferative lesion involves the upper portion of the right upper. The exudative lesion is seen below it as a soft cloudy shadow, in marked contrast to the harder, stringy lesion above. The exudation occurred suddenly, following parturition and, with bed rest, cleared completely in six months. The apical lesion remains unchanged.

weaknesses in the older classifications of pulmonary tuberculosis. They take no account of the quality of reaction. Irrespective of the quantity of reaction our therapy must be modified by the quality. Undoubtedly the great improvement in the treatment of pulmonary tuberculosis made in the last ten years is almost entirely due to better and more frequent x-ray studies of the chest. While it is true that the words "proliferative" (productive is better) and "exudative" do not adequately describe the difference between these two types of lesions, and while it is also true that both types may be present in the same individual, yet we recognize the predominant type from the x-ray picture and treat accordingly. The exudative lesion is the most dangerous and requires the most intensive and immediate rest care, but may offer the best results in the end. This type is most frequently seen in our young people and furnishes the start for what we termed in the past, "galloping consumption." These patients require immediate care, and if they have to wait for weeks or months to obtain a bed in a sanatorium may lose all hope for recovery. If they have no means of complete hospitalization or adequate bed rest in a home, they should be considered emergencies and never be compelled to wait; for within a few days they can advance from a minimal to an advanced tuberculosis. Their lesions should be described in classification.

While productive lesions also require care, such care need not be so immediate or so intense. When tuberculosis was treated on physical signs alone many patients with exudative tuberculosis died for want of diagnosis, and many patients with productive tuberculosis were retained in bed for years unnecessarily.

I note Doctor Shipman's experience with nurses, which seems to indicate that exudative type lesions give a greater tuberculin skin sensitiveness. This is a work which will bear further study and is of great importance. Our ideas as to allergy, resistance (or immunity) are still vague. There seems to be more in immunity than just allergy.

We see a type of pulmonary lesion in children which we call "allergic" for want of a better name. It differs from the adult exudative in appearance, but clears without x-ray evidence of scar as may the exudative. There may be several types of pathologic changes in what we loosely term an "exudative lesion," and this would account for the various results observed from complete resolution to part resolution

and part fibrosis, and even excavation. But whatever the basic pathology may be, we shall do well to keep in mind this x-ray classification, because of the clinical value.

✱

F. M. POTTENGER, M. D. (Monrovia).—The author's paper deals with an important subject in clinical tuberculosis. The older classifications, based on extent of the lesion, were unscientific and gave no basis for prognosis. They served a good purpose, however, in making men examine more carefully in order to determine how much disease was present, and were responsible for the development of much of the expertness in physical examination which has characterized the work in chest clinics in recent years. Rathbun's addition of the physiologic reaction to the anatomical extent as adopted by the National Association is a great improvement.

The only real basis for classification, to my mind, however, is the immunologic one. With a conception that the whole picture of clinical tuberculosis is either directly or indirectly produced by the patient's specific reaction to his infection, we have a basis for understanding every aspect of the disease. We cannot understand any of the phenomena which are present during its course apart from the immunity reaction.

It is our conception that the cell sensitization which causes the allergic reaction is a step on the way toward the establishment of immunity. As immunity becomes more effective the allergic reaction becomes less necessary and less evident. The higher the sensitization of the cells the greater the allergic reaction, and the greater the allergic reaction the more prominent the exudative phenomena. This is why the preponderantly exudative tuberculosis is more active and more dangerous than the preponderantly proliferative. When patients are ill for a long period of time it will be seen that the exudative phenomena are less prominent in comparison with the opportunities for reinoculation than they are in earlier lesions. There is, on the other hand, an increase in proliferative effects in those cases as they advance, even in those which were primarily exudative in character. This effect may be considered as being due to a marked desensitization to tubercle bacilli and bacillary protein, or, which is the same thing, to an increase in the degree of immunity present.

This desensitization particularly characterizes cases of tuberculosis which are preponderantly proliferative in type, the so-called fibroid cases. Cases are sometimes preponderantly proliferative from the very start, as mentioned by the author, and may remain so until healed; or until, for some reason, either an excess in dosage of bacilli or a depression of immunity, allergic phenomena again come to the fore, and cause an exudative reaction to be superimposed upon the proliferative process. The preponderantly proliferative lesion is relatively mild.

Since patients suffering from chronic proliferative tuberculosis do not show the same degree of toxemia as those with exudative lesions of similar extent, they can be treated differently. They can be put on exercise sooner and be treated by a less restricted regimen. There is one thing, however, that must always be borne in mind in considering treatment for the chronic proliferative type, and that is, that the toxemia is so slight that the patient too often finds himself with an extensive lesion before he knows it is present. He is usually apprised of its presence by a breakdown of his immunity, associated with an increase in allergic reaction, which is shown by an increased acuteness, or by dyspnea, which is produced by the gradual restriction in the amount of functioning pulmonary tissue, or by symptoms on the part of the gastro-intestinal tract. So for this reason the proliferative type of tuberculosis should be treated seriously as soon as recognized so that the patient be given full chance for recovery.

There is a tendency on the part of some clinicians to assume that small proliferative lesions are benign and require no special attention. When one recalls that all extensive proliferative processes were once limited in extent, the necessity of treating any lesion which is unhealed as a potential danger must be evident. It may be treated differently, but it must be followed carefully and the patient must be kept under rigid surveillance until healed. Doctor Shipman rightly makes the difference in character of lesion the basis for a variation in therapy, but he does not make the preponderantly proliferative process a basis for a let alone policy, as some are inclined to do.

✻

C. E. ATKINSON, M.D. (Southern Sierras Sanatorium, Banning).—The title of Doctor Shipman's article, "The Clinical Classification of Pulmonary Tuberculosis," appeals to me as having been exceptionally well chosen. Although exudation and proliferation are, as Doctor Pottenger states, essentially merely different phases of the same process, varying only quantitatively, yet for clinical purposes and as a working guide to prognosis and treatment, a classification along the lines suggested is most helpful.

I think it is understood that the classification should go further, including, so far as feasible, notations such as the extent and location of the lesions, and recording the presence or absence of cavities.

My own views coincide with the discussions by Doctor Bush and Doctor Pottenger. As pointed out by Doctor Shipman, exudative lesions at times clear rapidly; and in some instances no evidence, or practically no evidence of the tuberculosis, remains. We may well bear this fact in mind when reexamining a patient who has previously been informed by another physician that he has tuberculosis. If a reputable physician has previously made a diagnosis of tuberculosis, even though our findings at a later date are practically negative, we are not justified in telling the patient that he did not have tuberculosis. Most persons have the impression that tuberculosis always leaves scars or some reminder of its prior presence; so, if our examination discloses no evidence of tuberculosis, it seems a good plan to explain to the patient that exudative tuberculosis may disappear completely, with no trace remaining.

In cases of the proliferative type with cavitation, it is not uncommon for an acute bronchogenic extension of an exudative nature to occur in the oppo-

site lung. In a number of such patients, I have regretted not having adopted pneumothorax treatment sooner. Even in the presence of an exudative extension in the contralateral lung, if it is not too widespread, collapse therapy may still prove effectual.

Exudative tuberculosis is often brought to attention by a flu-like attack, or a so-called cold, and sometimes resembles a bronchopneumonia. It may develop rapidly into a veritable galloping consumption, and many cases will be altogether missed unless x-ray studies are made routinely.

Now and then there may, at an early stage, be difficulty, particularly as regards roentgen evidence, in distinguishing exudative tuberculosis from a non-specific congestion or inflammation, or from a more or less localized pneumonic process, such as may be produced by influenza. A careful review of the evidence from all standpoints is, then, necessary before arriving at a diagnosis.

Exudative tuberculosis demands a prompt and intensive application of rest, and extraordinary watchfulness is imperative. If a satisfactory response is noted, no further measures are required. Owing to the comparative thinness of the cavity walls and to the relative absence of fibrosis, cases of predominantly exudative tuberculosis with excavation yield exceptionally well to collapse therapy. Cavities sometimes close completely under the more ordinary regimen, but frequent check-ups should be made, and if the case is not responding satisfactorily, some form of collapse therapy, in the absence of contraindications, should be instituted without further delay.

BLOOD TRANSFUSIONS IN CHILDREN*

By PHILLIP E. ROTHMAN, M. D.
Los Angeles

DISCUSSION by P. F. McMurdo, M.D., San Francisco;
E. P. Cook, M.D., San Jose.

IT is very difficult at the present time to properly evaluate the status of blood transfusions in pediatrics. Reports by Sidbury and by Krahulik and Koch describe results in practically every disease of childhood. It is at once apparent that, despite the type of illness or the condition of the patient, transfusion is a fairly benign form of therapy free from the dangers formerly ascribed to it. Therapy, however, that is used simply because it will do no harm and may do some good, is obviously not scientific medicine and will quickly deteriorate to the level of many obsolete panaceas. The most careful statistical analyses are necessary before we can feel certain that mortality rates have been reduced. The purpose of this paper is to give a brief review of prevailing impressions concerning blood transfusions in children and to reemphasize some of the details of technique.

BLOOD DONORS

The amount of blood required for infants is comparatively small and, consequently, very little attention is paid to donors. Nevertheless, the blood of any individual who is repeatedly used as a donor should be investigated in order to avoid the production of an anemia. Although one person on record has been used sixty times and given 50,000 cubic centimeters of blood within six years (a total loss of ten times his blood

* Read before the Pediatric Section of the California Medical Association at the fifty-ninth annual session at Del Monte, April 28 to May 1, 1930.